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AMITRIPTYLINE INDUCED SIADH PRESENTING AS HYPONATREMIA

Medicine

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KEYWORDS

INTRODUCTION:

SIADH is characterized by hyponatremia and impaired urinary dilution (urine osmolality >100 mOsm/kg) in the absence of hypovolemia, hypotension or other non-osmotic stimuli to ADH release, Euvolemia, high urinary Na > 20 mEq/L, low serum uric acid, normal potassium, normal acid base balance, and no signs of hypothyroidism or adrenal insufficiency.

Basic pathophysiology is failure to mount water diuresis even when intake exceeds the renal and insensible losses[1]. Increase in ECF volume causes ANP release and decrease in aldosterone causing the natriuresis and isotonic loss of ECF, thereby causing euvolemia[1]. There are various causes of SIADH which include- Paraneoplastic SIADH, Neurological pathology, pulmonary pathology, Head injury, drug induced, genetic (nephrogenic SIADH)[2], etc. Drug induced SIADH is becoming more frequent cause of hyponatremia. Here we present a rare case of amitriptyline induced Hyponatremia in a 48-year-old female patient.

CASE REPORT:

A 48-year-old female presented to GMCH Nagpur casualty with complaints of abnormal behaviour for 4 days and not able to talk for 1 day. Patient was diagnosed case of systemic hypertension with Hypothyroidism with old cerebrovascular accident with Lumbar disc herniation (L3-L5) on treatment and following up in GMCH and SSH Neurology opd, Nagpur. Patient was admitted for evaluation and proper management. Patient was on following drugs

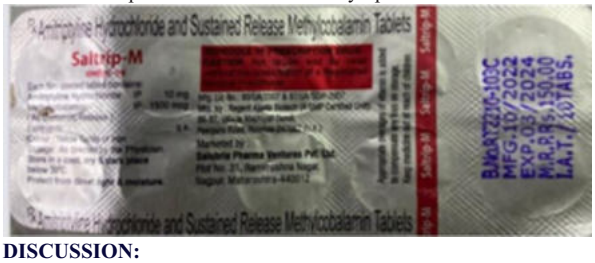
1. Tab Rasonel Asp 150+20 OD (Aspirin +Atorvastatin)
1. Tab Telmisartan 40 mg OD
2. Tab Thyronorm 25 microgram OD (Thyroxine)
3. Tab Saltrip-M 10 mg HS (Amitriptyline)

Patient was started on Tab. Saltrip M for radicular pain due to lumbar disc herniation since August 2021.

On examination, patient had BP= 130/90, Pulse= 96/min. patient was conscious, not oriented to time, place or person and not following oral commands. Respiratory, cardiac examination was normal. Per abdomen was soft with no organomegaly. Patient had no focal neurological deficit, bilaterally plantar reflex was flexor, DTRs were ++ in all 4 limbs. Patient had lead pipe rigidity in all muscle groups of all limbs. MRI brain was suggestive of chronic infarct in left hemi pons and left corona radiata along with chronic small vessel ischaemic changes in bilateral centrum semiovale. USG Abdomen was normal. CSF analysis was done which had proteins- 36 mg/dl, sugar 81 mg/dl, cells= 31/mm^3, 100 % lymphocytes. TFT was done which had T3= 0.8 ng/ml, T4= 14.1 microgram/dl and TSH= 1.7 microIU/ml. Serum Sodium was found out to be 119 mEq/L. Other laboratory investigations were done and are shown in the TABLE 1.

INVESTIGATION ON ADMISSION	VALUES
HEMOGLOBIN	9.9 g/dl
TLC	10500/mm^3
PLATELET COUNT	514000/mm^3
S. UREA	26 mg/dl
S. CREATININE	1.2 mg/dl
S. SODIUM	119 mEq/L
S. POTASSIUM	4.1 mEq/L
TOTAL PROTEIN	5.9 g/dl
TOTAL BILIRUBIN	0.5 g/dl
ALP	98 U/L
AST	21 U/L
ALT	14 U/L
S. ALBUMIN	3.6 g/dl
TRIGLYCERIDES	57 mg/dl
T. CHOLESTEROL	115 mg/dl
HDL	61 mg/dl
LDL	43 mg/dl
SERUM AMMONIA	74 microgram/dl.
URINE SODIUM	64 mEq/L
URINE POTASSIUM	27.3 mEq/L
URINE CHLORIDE	82 mEq/L
URINE OSMOLALITY	447 mOsm/kg H_2O
SERUM OSMOLALITY CALCULATED	250 mOsm/kg H_2O
SERUM CORTISOL	14.26 microgram/ dl

Patient was started on Inj 3% NS 100 ml @ 20ml/hr. Tab. Saltrip M was withheld. Patient improved over 24 hours. Patient was conscious and fluently communicating and following all commands. On examination, normal tone was found in all 4 limbs. Serial serum sodium levels were done which were as follows; 119 mEq/L. Patient was followed up after 1 week and had no symptoms at all.



DISCUSSION:

Hyponatremia is a clinical complication of a wide variety of diseases, surgical procedures, and drug treatments, when defined as plasma sodium concentration of less than 135mEq/L. Hyponatremia induces generalized sweeling, a consequence of water movement down the osmotic gradient from hypotonic ECF to the ICF. Early symptoms of hyponatremia include nausea, vomiting and headache. However severe symptoms my evolve rapidly which includes seizures, obtundation, brainstem herniation, coma and even death.

Hyponatremia is a recognized side effect of antidepressant medications[3,4].

Hyponatremia due to anti-depressants develops in a wide range of 3 to 120 days of treatment initiation and is usually reversible between 2 days to 1 month of withholding the SSRI[5]. Risk factors implicated for the development of hyponatremia include old age, female gender, and a low BMI[5]. SSRI have been shown more frequently associated with hyponatremia than other antidepressants[6,7]. Amitriptyline induced hyponatremia is rarely reported[8]. Most cases of antidepressant induced hyponatremia occur in elderly people, which could be related to altered ADH regulation or its action on kidneys[9]. But in this case

hyponatremia is observed in young female patient. So, clinicians and especially psychiatrist should be vigilant while prescribing patient with antidepressants in elderly as well as young people.

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